

220

A COMPREHENSIVE STUDY OF NONINFECTIOUS NEUROLOGICAL
DISORDERS OF BOVINE

by

655
G. NARASIMHA MURTHY

B. V. Sc., Madras University, 1943

A MASTER'S REPORT

submitted in partial fulfillment of the
requirements for the degree

MASTER OF SCIENCE

Department of Medicine and Surgery

KANSAS STATE UNIVERSITY
Manhattan, Kansas

1964

Approved by:


Major Professor

LD
2668
R4
1964
M 984
C. 2

TABLE OF CONTENTS

INTRODUCTION	1
CONGENITAL AND/OR HEREDITARY NEUROLOGICAL DISORDERS	4
Cerebellar Hypoplasia and Degeneration	5
Congenital Ataxia	6
Cerebellar Agenesis	10
Spastic Paresis	11
Congenital Posterior Paralysis in Calves	12
Spastic Syndrome in Cattle	13
Epilepsy	15
Spina Bifida	16
Hydrocephalus	17
Bull Dog Calves	21
NONINFECTIOUS NERVOUS DISORDERS DUE TO ENVIRONMENTAL AND EXTRENSIC FACTORS	22
Lightning Stroke and Electrocution	22
Heat Stroke, Heat Exhaustion, Heat Cramps and Dehydration Exhaustion	24
Heat Stroke	25
Sun Stroke	26
Heat Exhaustion	27
Heat Cramps	28
Trauma	28
Diseases of Peripheral Nerves	32
Allergic Encephalitis	32
INTRINSIC FACTORS	33

Tumors	33
Symptomatic Disorders	37
NERVOUS DISORDERS IN BOVINES DUE TO NUTRITIONAL DEFICIENCIES	39
Vitamin A Deficiency	39
Thiamin Deficiency	41
Mineral Deficiencies	41
NONINFECTIOUS NEUROLOGICAL DISORDERS DUE TO POISONOUS PLANTS AND CHEMICALS	45
Strychnine	46
Loco Poisoning	47
Vetchlike Astragali	47
Claviceps paspali	47
Belladonna (Deadly Night Shade)	48
Hemlocks	48
Lathyrus Poisoning	49
Snake Bites	52
Tick Paralysis	52
Lead Poisoning	53
Mercury Poisoning	54
Chlorinated Hydrocarbon Poisoning	55
Organic Phosphate Poisoning	56
NONINFECTIOUS NEUROLOGICAL DISORDERS OF UNKNOWN ETIOLOGY IN BOVINE	57
Polio-encephalomalacia of Cattle	57
Arthrogryposis and Hydranencephaly in New Born Calves	59
Neuronopathy and Pseudolipidiosis in Aberdeen Angus Calves	60
SUMMARY	61

ACKNOWLEDGMENTS	65
REFERENCES	66

INTRODUCTION

Neurological disorders of domestic animals have not been as completely studied as compared to those of man.

Many aspects of human nervous system have been studied because of the value of human life, and neurologists have better opportunities to explore the details of varied conditions with divers diagnostic aids such as electroencephalograms.

The veterinarian deals with different species of animals from the smallest mammal to the largest terrestrial creature the elephant besides the variety of avian species.

There are very many serious handicaps which contribute to the lacunae of shortage of knowledge about nervous disorders of animals.

The central nervous system is cased in a bony framework and is extensive. It escapes visual observation. Because of the extent and complicated structure and disposition, the clinical signs evidenced cannot be readily interpreted in its relationship to the pathological lesions.

The nervous symptoms are noticed in many secondary conditions--such as toxæmias, septicæmias, hyperthermia, viral and bacterial infections, plant and chemical poisonings, intoxications due to indigestion, degeneration of liver and metabolic disorders such as milk fever and ketosis--etcetra.

The clinical symptoms manifested by different disorders and diseases are objective in character, unlike the human who is endowed with power of speech and expression. It is often hard to correlate the clinical signs with the morbid conditions in the animal body. Many disease conditions

translate to clinical signs which are apparently similar.

The information available in textbooks on the subjects of veterinary neuroanatomy, neurophysiology, embryology, and pathology is incomplete.

It is observed from the literature, that a good deal of work has been done on small laboratory animals such as rats, guinea pigs and monkeys concerning the neurological disorders.

Little work has been done in the study of neurological disorders of the bovine, because the nervous disorders are surprisingly few, when compared with the disorders of other systems and also because of the labor involved to study the brain and spinal cord in post-mortem examinations. Many sporadically encountered nervous disorders express themselves with insignificant clinical signs. Many symptomatic nervous signs exhibited are secondary and may be due to disorders of systems other than the nervous system.

Very little information regarding the toxicity of many plant poisons and some chemicals which act on nervous system is recorded.

Much less information is available regarding the noninfectious neurological disorders in larger animals, which are often singular and sporadic. The veterinary clinicians and pathologists have been more engaged with the investigations of contagious and infectious disease in view of the frequency of their incidence and of greater economic losses. The materials for investigations can be easily procured for the purposes of research on the side of infectious and contagious diseases.

The farmers and dairymen have to assist the veterinary and animal husbandry experts by furnishing the complete history of the individual animals or herds, to arrive at a correct diagnosis and evaluate their

hereditary dispositions. The congenital and hereditary disorders can be studied only when the herd registers and records of individual animals are precisely maintained. Many details are sometimes lacking for tracing the geneology of animals, and assessing the hereditary defects.

In recent years many disorders of hereditary and of unknown etiology are being reported and investigated.

Before dealing with the subject of neurological disorders in the bovine, it would be necessary to discuss briefly the functions of central nervous system.

Functions of Cerebrum. The cerebrum is the seat of psychic functions, such as thought, feeling and will. Together with the pyramidal tracts, and extra pyramidal motor system, it constitutes the central motor system concerned with the voluntary movement and body sense, as denoted by skin sensation, joint and muscle sense, muscle tonus and various reflexes. In cerebral disorders, symptoms of disturbance of consciousness, motor irritation, spasms, convulsions, paresis, disturbances in cutaneous sense, and reflexes, disturbance of vision and hearing are produced (Gibbons, 1963).

Cerebellar disorders include disturbance in voluntary movement, concerned with equilibrium--falling backwards, incoordination of movement, and nystagmus. Cerebellum is part of motor system concerned with maintenance of muscle tone, steadiness of tone. Effects of cerebellar disease depends on the part affected and quantity of damage. Compensation occurs to a remarkable extent in damaged conditions. Hence the loss of purkinje cells in a single folium or more in animal cerebellum does not have the significance to exhibit clinical signs (Innes and Saunders, 1957).

Other symptoms include muscular weakness and marked opisthotonus. In

unilateral lesions the signs are restricted to one side of the body.

The spinal-cord and peripheral nerves are concerned with transmission of impulses, and in control of organs having involuntary musculature. Disorders of this part are manifested by paresis and paralysis.

For purposes of convenience the noninfectious neurological disorders are discussed under the following classifications:

Noninfectious neurological disorders due to

- (1) Congenital and/or hereditary defects,
- (2) Environmental and extrinsic factors,
- (3) Intrinsic factors,
- (4) Nutritional deficiencies,
- (5) Toxic plants and chemicals, and
- (6) Unknown etiology.

CONGENITAL AND/OR HEREDITARY NEUROLOGICAL DISORDERS

The study of hereditary conditions requires a sound knowledge of genetics, and congenital disorders requires a thorough knowledge of embryology.

For the study of hereditary conditions, the stock owner has to provide herd records for the analysis of their genealogy. If the genetic defect could be established by the relevant records, elimination of undesirable characters is possible.

Congenital defects are defects which originated in the early embryonic life.

In embryonic life, the first sign of embryo in the blastodermic

vesicle is the primitive streak.

The neural groove which is the precursor of nervous system is formed immediately behind the primitive streak. The great majority of congenital defects, common in calves, appearing as monstrosities, can be traced to defective development of nervous system. Failure of neural groove to close may account for double monsters, and spina bifida.

Failure of development of posterior neural pole causes a monstrosity in which the hind legs are deformed and rigid. Failure of the anterior pole to develop properly results in hydrocephalus cleft palate and Bulldog calves.

Cerebellar Hypoplasia and Degeneration

A familial form of cerebellar hypoplasia and degeneration in pedigree hereford cattle was studied by Innes et al. (1940) in England, where only a small number of breeding bulls were retained for a long time, which resulted in both inbreeding and line breeding. Certain cows mated with known bulls gave birth to paralyzed calves with a familial tendency. The calves were reported to be deficient in cerebellar development and did not improve in spite of treatment.

Animals were unable to rise, and retained lateral recumbency with neck and limbs stiffly extended. Opisthotonus and nystagmus were present with head tremors. The animals were conscious of the environment.

It was reported that, grossly the cerebellum was very small, and all the folia showed wasting. On section the texture was leathery and tough. The brain stem and cerebral hemispheres were normal superficially, as well

as in section. No inflammatory changes were present in leptomeninges. Lesions were confined to the cerebellar cortex. The cerebellar folium showed atrophy and absence of purkinje cells. The cerebellar cortex showed large neuroblasts, in the molecular layer, and a few in granular layer--suggestive of arrest in development. The cerebral cortex showed gliosis in the molecular layer which is also suggestive of a degenerative process. Degenerative changes in purkinje cells and other neurons were prominent.

A similar condition was reported by Young (1962). It is stated that three calves affected with cerebellar ataxia were born in a herd of 80 crossbred Angus-Shorthorn cows. Signs of disease were noticed from the time of birth and no gross abnormalities were noticed in the brain and spinal cord.

Histological studies revealed a depletion or absence of myelin in the cerebellum, pons and medulla and other parts of central nervous system. Gliosis accompanied these changes in some areas. Breeding records were not adequate to study the hereditary disposition.

Another similar condition in a Holstein-Friesian calf with identical lesions were described by Anderson and Davis (1950).

Congenital Ataxia

This condition has been reported in Jersey calves and Angus-Shorthorns.

Saunders et al. (1952) reported that 23 calves born in 15 years in a purebred Jersey herd showed the following signs.

Symptoms were noticed at the age of one to two weeks or at birth. In calves that showed symptoms at birth, the joints were stiff in the front legs

and the limbs were hanging straight from shoulders. The left front leg was affected in all animals. Both sexes were affected. Nystagmus was not noticed. No disorder of muscle tonus was observed. Cutaneous sensibility to pain was present. The animals were responsive to audio-visual stimuli. No evidence of cranial nerve disfunction was present.

Clinically the disease appeared as an ataxia or failure of functionally associated muscle groups to act in harmony.

Gross pathological lesions were minimal. Histopathological examination revealed loosely reticulated feltwork occupying the white matter of the cerebellum and running into the medullary core of each folia in all lobes. Oedema fluid was present in the interstices of the meshwork. Similar lesions were present in the medulla. No lesions were noticed in the thalamus, hippocampus, basal ganglia, cerebral cortex and spinal cord. The subcortical white matter was oedematous in the cerebral sections. Some of the meshwork consisted of nerve fibers devoid of myelin sheaths, especially towards the periphery of the medullary cores of cerebellar folia. In addition to large areas lacking in myelin, and paucity of nerve fibers, a diminution in the number of nerve cells was apparent in certain areas of brain. There was no evidence of gliosis anywhere in the brain. Most of the pathological process was established at birth.

The signs of incoordination and failure of synergistic muscle groups to act in harmony were similar to those of congenital cerebellar hypoplasia described earlier. The cerebellar cortex is histologically unchanged in structure and no disintegrating myelin is present.

The condition was a congenital hereditary disorder. The affected offspring was born from phenotypically normal parents, and the disease is due

to a recessive gene. Since calves of either sex were affected, the character is not sex linked.

Hereditary congenital ataxia of a similar nature has been reported by Johnson et al. (1958) in Holstein-Freisian calves.

A total number of eight calves showing spastic symptoms were found in two related herds of Holstein-Freisian cattle over a two year period.

Symptoms were those of incoordination, a failure of synergistic muscle groups, to act harmoniously. The symptoms were similar in all cases and appeared at birth. Calves of either sex were affected (not sex linked). An analysis of pedigree record of calves revealed one common ancestor. This bull appeared in the pedigree of both sire and dam of all affected calves. The ataxia is postulated to be inherited as an autosomal recessive. The occurrence of ataxic calves ceased when the bulls known to be carriers were removed from the herd.

The anatomical condition was neuronal degeneration. Since the affected offspring were born from phenotypically normal parents the disease if inherited would have to be due to a recessive gene.

Hulland (1936) studied a similar condition with identical symptoms of lesions in a purebred Shorthorn breed and in purebred Herefords. It was described to be a hereditary condition, and recessive in character.

Coles and Moore (1942) have reported similar congenital spasms, as a recessive lethal condition in Holstein-Freisian calves. The calves were unable to rise or stand unaided, and showed constant tremors of skeletal muscles, both when recumbent or held upright. The histopathology was not described.

A similar type of congenital hereditary spasms in Jersey breed has been

described by Gregory et al. (1944).

The affected calves exhibited continuous, intermittent spasmodic movements of the head and neck, usually in a vertical plane. When the animals were forced to stand, spasms in both front and hind legs, hampered standing and walking. The spasms were not intensified or initiated by noise or shock. Even though the animals had a good appetite, they died in a few weeks after birth.

The spasmodic condition was ascertained to be inherited as a recessive autosomal lethal. All the herds in which the defective calves appeared were using closely related bulls, purchased or descended from a single herd of high producing purebred jerseys.

In both the conditions of (1) cerebellar hypoplasia and (2) congenital ataxia, incoordination, failure of synergistic muscle groups to act harmoniously, stiffness of neck and limbs and unimpaired consciousness are common symptoms.

In hypoplasia with degeneration, the cerebellar cortex is atrophied, thick and leathery while in congenital ataxia, the cortex is not affected and is normal in size and texture, and only the white matter was affected. The cerebellar cortex, in hypoplasia with degeneration exhibited large number of neuroblasts in molecular layer, and a few in the granular layer, and a gliosis of molecular layer suggestive of degenerative changes which are totally absent in ataxia. In cerebral ataxia, lack of development of nerve cells, axon cylinders and myelin sheaths and oligodendroglia are noticed in the white matter of the cerebellum, thus presenting a reticulated spongy appearance. Lesions in the white matter extend to cerebellar folia, cerebral cortex and thalamus. In cerebellar ataxia, there is no disintegration of

myelin; and gliosis is absent.

Congenital ataxia is conclusively proved to be a hereditary condition with autosomal recessive gene.

Both conditions are incurable.

The lesions and symptoms described are insufficient to distinguish cerebellar hypoplasia, and congenital ataxia. Analysis of heredity is conclusive. The conditions were therefore described under confusing titles--eg. congenital lethal spasms, etcetera.

Cerebellar Agenesis

This is a type of hypoplasia of the cerebellum grossly evident in new born calves and other animals. The clinical signs described are those of cerebellar ataxia. These cases are often sporadic, and very few instances are on record. The etiological factor is suspected to be familial.

Johnson et al. (1962) have reported cerebellar hypoplasia in one Short-horn with Hereford cross, one in Shorthorn, and one in Hereford stocks.

The three calves were not able to stand from birth, and one lay in lateral recumbency. The limbs were held stiff in extension. In two cases the cerebellum was completely absent, only represented by cerebellar peduncles, and in the remaining one, the cerebellums weighed 10 grams of a total brain weight of 197 grams.

Three more cases of cerebellar agenesis have been described by Sholl et al. (1939), wherein they stated that there was a total absence of cerebellum, or a gross reduction in size of all lobes in some cases.

Even though it is seen to be a congenital disorder, it was not

established to be hereditary.

Spastic Paresis

Spastic paresis of cattle has been reported by Formstan and Jones (1956). A similar condition was reported by Innes and Saunders (1957) in Holstein-Friesian cattle.

The disease has been described to occur in calves two to six months old, and occasionally in older cattle. The condition is characterized by excessive rigidity of one or both gastrocnemius muscles, often unilateral, and developing only a few weeks after birth. The condition progresses to a paresis of the affected limbs. No indication of paralysis of bladder or rectum is noticed.

Symptoms. Marked spasms of the quadriceps and gastrocnemius muscles with rigidity of the tendoachilles are observed. The spasm is noticed more pronounced when getting up. Marked abduction of the limbs is noticed, with the toe only touching the ground. The affected limb swings like a pendulum when the animal walks.

No inflammation or enlargements of joints or patellar dislocation has been reported. The patellar reflex is exaggerated. Histologic investigation revealed no abnormality in the brain and spinal cord. It is believed that the disease is inherited as an autosomal recessive condition.

A similar report wherein the fore limbs were affected was published by Galist and Chichini (1961).

The syndrome is characterized by spastic contraction with hypotrophy of muscles of the forearm with predominance of flexors of the metacarpus,

resulting in marked outward and forward flexion of the carpus. The muscular spasm was either intermittent or permanent and may be uni or bilateral.

The etiology has been concluded to be as a hereditary factor, since the calves had descended from a bull whose progeny had spastic paresis of hind limbs.

Congenital Posterior Paralysis in Calves

This condition was reported (treated as a separate syndrome) by Innes and Saunders (1957) in Red Danish Cattle.

The calves exhibited complete posterior paralysis of hind limbs. They exhibited opisthotonus, and some suffer from continuous muscular tremors. Some developed keratitis in three days after birth. This condition occurred as a hereditary disease in Norwegian Red-poll breed, and the mode of inheritance was said to be recessive. No central nervous system lesions could be detected.

A similar condition in Danish breed with a recessive hereditary defects has been reported by the same authors.

The calves remained recumbent at birth with the legs fully extended. The joint and tendon reflexes were fully accentuated. The muscles of hind legs were spastic. The musculature and peripheral nerves were not diseased as evidenced by electromyography. The increased reflex tonus was of an extra pyramidal nature, which was conformed by prominent degenerative changes in globus pallidus and reticular formation.

Spastic Syndrome in Cattle

This condition has been reported from Switzerland, Germany, and the United States in cattle in dairy breeds.

The disease is characterized by intermittent spasticity of muscles of rear limbs or the whole body in advanced cases.

It is being called as "Crampy," "Neuro-muscular spasticity," "posterior paralysis," "spastic syndrome," "progressive posterior paralysis" and "stretches."

It is observed by Becker et al. (1961) that the genetic influence on crampy was conclusive, since the condition occurred in cattle with closely bred family lines and did not occur in other closely bred and unrelated lines. The character developed in animals where both sire and dam are affected. It is possible that multiple gene inheritance may be involved. The condition is inherited as a qualitative characteristic. The condition is neither sex linked, nor sex limited. It is not a dominant character since it frequently skips generations in expressing itself. Inheritance appeared to be a single recessive factor with incomplete penetrance.

It is a slowly progressive disease extending over a period of years. The incidence is reported more in Holstein, Guernsey, and Ayrshire in order of frequency and has been observed in the Angus breed (Gibbons, 1963). The disease appeared in both sexes with no respect to season. Cattle above three years of age alone were affected and more commonly in cattle aged six years or more.

It is believed to be a functional disease of the central nervous system.

The cramps or spastic contractions initiate in one or both hind legs

and later extend to the back. The cramps in early stages last from several seconds to a few minutes and cease abruptly and repeated on stimulation. Attacks which were mild initially progress to severity in course of years. Attacks may be separated by days or weeks in the beginning but become frequent with the progress of the disease.

Symptoms are absent when the animal is recumbent, but are evident when the animal first gets up or when excited.

No histopathological changes are observed or reported in the central nervous system. No gross lesions can be detected since the disease is of an intermittent character.

Spondylitis and disc lesions in the lumbo sacral regions have been observed by Martin (1959).

The first symptoms are mild and are often observed late in the course of the disease. Symptoms are observed when first getting up or when the animal is excited.

The animal will extend or flex one hind leg (more often the left), as the muscles of limb go into continuous spastic contraction that lasts for a few seconds to a minute and then suddenly ceases. After a pause the spastic condition repeats.

As symptoms progress, both rear legs are affected. In the later stages the muscles of the back are involved. When both rear legs are involved, and when the animal is driven forward, the animal walks on the toes with a spradling stilted gait with the tail quivering, and sweats profusely due to pain.

In advanced stages the attacks are frequent. There is weight loss, and milk production is decreased. Bulls are unable to mount cows. During the attack the animal is unable to lower the head to eat or drink. The lumbar region is depressed. Consciousness is not impaired at any stage. External stimuli such as noise, fright, physical exercise aggravates the condition.

In severe spasms, tremors of groups of muscles first

appear over the body. The symptoms suggest that there is some damage of postural reflex mechanism which include muscle tonus, standing reflex, attitudinal or stance reflexes (Roberts, 1953).

Epilepsy

Epilepsy is a sudden brief or prolonged loss of consciousness usually preceded by a convulsion.

Symptomatic epilepsies are traceable to a discoverable disease process such as trauma which produces localized lesions in the central nervous system.

Symptomatic convulsions due to trauma, poisoning, cerebrospinal nematodiasis can be mistaken for true epilepsy.

Idiopathic or true epilepsy is a convulsive state, which cannot be associated with a known etiologic factor or definite underlying lesions. Animals are perfectly normal between attacks. The condition persists for the life of the animal.

Some fits may be preceded by a local motor phenomenon, such as tetany or tremor of one limb or the face. The convulsion may spread from this initial area to the rest of the body. This form is referred to as Jacksonian epilepsy. The attacks are always recurrent and the animal is normal in intervening periods (Blood and Henderson, 1963).

The condition is neither preventable or curable.

Spina Bifida

Spina bifida (cleft spine) is a congenital anomaly of spine characterized by incomplete fusion of the laminae to form the vertebral arch. This condition has been reported in humans, dogs and sheep beside cattle and rabbits.

This condition was reported in cattle by Epstein (1955), Goss and Hull (1939), Nes (1959), and McFarland (1959).

McFarland (1959) has described a typical case of spina bifida with a meningocele in a calf. Wherein it is stated that the duramater, herniated through the laminar defect, as a sac filled with cerebrospinal fluid. The calf was Angus male eight days old. The hind limbs were flexed and the anus prolapsed. Radiograph showed incomplete fusion of the neural arches of the last two lumbar and sacral vertebrae. The fore limbs showed deformity. The muscles of hind limbs were atrophied, and were yellowish white instead of reddish brown except gracilis Sartorius and adductor muscles.

The duramater was involved in the skin defect of five centimeters at the region of tubersacrale. The lesion was devoid of hair, and fluctuated on palpation.

Within the cavity at the level of fourth lumbar vertebrae, the spinal cord terminated in an atypical cauda equina. Conus medullaris was absent and the terminal nerve fibers coursed in a different direction. Some nerves passed through intervertebral foramina, while others passed dorsally into superficial tissues. The coccygeal muscles were atrophied, and the tail hung limp.

No abnormalities were described in the digestive, urogenital and

circulatory systems. Laminar fusion from the fifth lumbar to the fifth sacral vertebrae was absent. The spinal cord showed a typical structure with no pathological alterations, but nerve bundles of the sciatic nerve showed axon degeneration. The pathological muscle tissue was devoid of muscle fibers between the normal and pathologic muscle fibers.

The embryology of spina bifida was explained to be due to (1) a primary defect, involving the mesoderm, such that the laminar bone is not formed but replaced by connective tissue and (2) a primary defect involving the neuroectoderm, such that it suffers from dysplasia and secondarily induces a failure in laminar fusion.

Both heredity and environment have been incriminated in this anomaly.

The cases described by Goss and Hull (1939) are suggestive of a hereditary origin. The fault lies in the formation of the neural tube only.

Hydrocephalus

Hydrocephalus is the slow accumulation of excessive cerebrospinal fluid in the lateral and other ventricles and sometimes in the subarchnoid spaces due to obstruction of the lymph drainage.

Normally there is a continuous secretion of fluid from the choroid plexus of vessels located principally in the lateral ventricles. This drains through the aqueduct of Sylvius into the fourth ventricle. Then it passes through some minute openings in the roof of fourth ventricle, known as foramina of Luschka into basal cisterns in the subarchnoid space. A small quantity of cerebrospinal fluid finds its way into the spinal canal. The overflow of the fluid from the basal cisterns anteriorly, through the

intercommunicating subarchnoid spaces gets reabsorbed into venous circulation through arachnoid villi which project into the venous sinuses.

Hydrocephalus is generally believed to be caused by some mechanical obstruction in the cerebrospinal fluid pathway obstructing the outflow of lymph. It may occur due to excessive secretion of the fluid or defective absorption to the subarchnoid villi.

The vulnerable seats of obstruction of the cerebrospinal fluid pathway are the aqueduct of Sylvius and foramina of Luschka which leads from the fourth ventricle.

Hydrocephalus is spoken of as congenital when there is an embryonic defect in the drainage canals and foraminae in between the ventricles and subarchnoid space. In acquired hydrocephalus, the obstruction of drainage is due to some space occupying lesion or inflammation.

Thus the causes of hydrocephalus may be due to developmental defects, haemorrhages, inflammation, thrombosis, tumors or cysts in cerebral hemispheres, third ventricles, fourth ventricle, diffused growths in leptomeninges, deformities in the base of the skull as achondroplasia, and spina bifida. It may also occur due to stenosis by pressure from exterior on the foramina of Munro or septum formation at the foramen Megendie, or Stenosis of aqueduct of Sylvius.

The clinical symptoms of hydrocephalus may either be due to (1) developmental defects, or (2) hypovitaminosis in calves due to failure of growth of cranial vault resulting in pressure on the growing brain, or (3) pressure from growth such as tumors or (4) functional failure of choroid plexus or subarchnoid villi.

Symptoms are developed gradually; there may be depression, disinclination

to move, a vacant stare, incoordination in movement, half closed eyes.

Cutaneous stimulation is reduced.

In some cases, charging, head pressing tremors and convulsions appear, finally ending in paralysis.

In case of hypovitaminosis, blindness and papilloedema are noticed earlier, and later the other symptoms of hydrocephalus develop.

In any case the cerebrospinal fluid is not altered biochemically or cytologically, but the cerebrospinal fluid pressure is increased.

In the congenital form of hydrocephalus, there is some malformation of the brain which interferes with fluid circulation through the ventricles and subarchnoid spaces. In early foetal life, the ventricles and choroid plexus are relatively large and the cerebral covering is thin. Any obstruction at this foetal stage of outflow will then exaggerate an already marked physiologic state and prevent the formation of cerebral gray and white matter. Whatever may be the cause, the end result hydrocephalus is the same. The cerebral hemispheres undergo pressure atrophy from within and/or without, and may eventually be reduced to thin walled sacs filled with clear or turbid fluid. The frontal and parietal bones bulge outward and the skull assumes a huge dome shaped edifice. The earlier the process starts before advanced cranial bone formation, the greater the cranial distortion. It may be lethal at birth or shortly after. There may be reduction in the orbital cavities. The convolutions above the dilated cavities become flattened. The choroid plexus may present a gelatinous appearance, but in adult animals (acquired hydrocephalus) with fully sutured skulls, there may be no cranial deformity and there may be only just marked dilatation of the ventricles (Innes and Saunders, 1962).

Congenital hydrocephalus has been reported by many workers. Cole and Moore (1942) have reported congenital hydrocephalus, described as an inherited variety, in Holstein-Freisians, the mode of inheritance being recessive.

Blackwell et al. (1959) reported hydrocephalic cases in calves which were stillborn or died soon after birth.

The skull was dome shaped and teeth development incomplete. Some births were accompanied by excessive amounts of amniotic fluid. From the herd history it was concluded, that the condition was due to a single autosomal recessive gene.

Baker et al. (1961) described cranial malformation in calves which occurred during early embryonic life and differed from the type of congenital hydrocephalus resulting from blocked drainage of cerebrospinal fluid.

In a group of 40 cows, all of which had given birth to affected calves, 20 were mated to a known transmitter of the condition, the remaining 20 to another bull of known ancestry without a history of hydrocephalus. The following breeding season the bulls were interchanged within the two groups. It appeared conclusively that the condition was inherited by a single autosomal gene.

Another interesting case of subdural hydrocephalus in a calf was reported by Williams and Frost (1938).

A Holstein-Freisian calf with a hydrocephalic sac, anterior to cranium was destroyed and the autopsy findings were noted. The palatine bone and vomer were absent. The nostrils were widely separated and opened laterally. The superior border of the ethmoid was deflected from its normal course by pressure of the hydrocephalic sac. The hydrocephalic sac was continuous with the intracranial duramater and protruded through the enormously enlarged persistent anterior neuropore. The nasal portion of the frontal bone was absent, while the cranial part of it was well formed. The persistent neuropore was bounded dorsally by the frontal bones and ventrally the basic ethmoid bone. The brain was compressed and dwarfed. The cerebrum was relatively thick. The hydrocephalic fluid had pushed the brain away

from basioccipital, sphenoid and ethmoid bones. An excess of fluid had pushed the dura and pia mater wide apart throughout the cranial cavity.

The defect here was quite different from ordinary hydrocephalus in which the fluid filled the lateral ventricles. Here the anterior neuropore persisted in the cranial walls and duramater remained open, leaving the brain naked.

The primary defect was a persistent anterior neuropore in the embryo.

This shows the defective development in the embryonic stage.

Bull Dog Calves

The condition described as bull dog calves by Berger and Innes (1948) and Gregory et al. (1942) under the title Achondroplasia denotes only developmental defects. The name bull-dog applied to aborted or stillborn monsters which resembled bull dog in the formation of the face. There was a shortening of the face due to arrested development of the nasal bones and maxillae. Many cases are aborted early but some reach full term to cause dystocia. Faetal anasarca, hydramnios, shortening of the limb bones, a round head bulging and a depressed short broad nose, a protruding tongue, a cleft or absent palate, a bifid epiglottis, a short stout neck were characteristic. Dwarfism occurs.

This condition is also traced to be an inherited neurologic disorder as a simple recessive character.

Achondroplasia (where there is imperfect ossification within the cartilage of long bones) disease starting in faetal life and producing dwarfism in humans similar to "Bulldog" calves has been recorded.

NONINFECTIOUS NERVOUS DISORDERS DUE TO ENVIRONMENTAL AND EXTRENSIC FACTORS

Lightning Stroke and Electrocution

Exposure to high voltage electric current in the form of lightning stroke or electrocution causes sudden nervous shock with temporary unconsciousness or immediate death. Residual nervous signs may persist after recovery from nervous shock.

An interesting symposium regarding lightning has been published by Morten et al. (1930).

The incidence of lightning stroke is high during summer months. Lands containing high quantities of iron salts are supposed to be more frequently affected. Trees, fences, barns, and pools of water are frequent attractions for lightning. It is said that oak trees, elm, conifer, beech and willow which are rich in starch and poor in essential oils are more attractive for lightning. When the flash falls, whatever assists the contact of electricity above, with that below, will receive some of the discharge, and the human being or animal, which have helped to complete the circuit will be affected. Large groups of animals exercise a strong attractive influence to the electric fluid.

Faulty wiring, high amperage, moist flooring, and earthing to pipe lines results in hazards of electrocution. Alternate current is more lethal than direct current.

Death is due to paralysis of the medullary centers with accompanying loss of complete consciousness and flaccid paralysis. Focal destruction of

nervous tissue occurs, and residual damage to the nervous system persists even after the shock disappears. Superficial burns may be evident at the site of contact with the current or along the path of flow from the point of contact to the ground. The burn is produced by the heat generated from the resistance of tissues to the passage of electricity.

Symptoms vary with the severity of shock. With high voltage current and good earth contacts, especially moist grounds, the animal dies instantaneously without any evidence of struggling. Singeing of the hair and burning may be evident if the animal is struck directly, but animals electrocuted by standing on electrified earth, do not exhibit marks of burns on the body (McCornell, 1946). In animals that have died of severe shock, unchewed hay may be found clenched in its mouth; bloody foam appears from the mouth and nostrils. Rigor mortis appears quickly and soon disappears. Decomposition sets in rapidly. Blood is tarry and noncoagulated, as death in electrocution is due to respiratory failure.

Burning may be superficial, but not under the skin in some cases, however in many cases there is bruising under the skin. Lacerations and fractures are more due to fright in animals, due to jumping, or falling in ditches.

There may be evidence of muscular relaxation, pupils are dilated, anal sphincter is relaxed and faeces passed at movement of death.

The burns may be localized to the muzzle or feet and be in the form of radial deposits of carbon with or without disruption of tissues, or they may appear as treelike, branching patterns or singeing running down the trunk and limbs. Burns from the head extend down the jugular furrow and fore feet to the ground, or from the croup to the ground along the hind limbs.

In less severe shock, the animal falls unconscious. Struggling is commonly seen followed by a period of unconsciousness varying from several minutes to several hours. When consciousness is regained the animal may return to complete normalcy or show depression, blindness, posterior paralysis, monoplegia, and cutaneous hyperaesthesia. In some cases nystagmus and unilateral paralysis are also noticed. Sloughing of the skin may occur after a few days. These signs may persist or disappear in a couple of weeks. The rectum and bladder are not paralyzed.

With minor shocks, the animal may be knocked down or remain standing. Consciousness is not lost. The animal shows restlessness, periodic convulsions of short duration followed by recovery after some days. Laboratory examination is not helpful in diagnosis.

Rigor mortis sets in and passes off quickly. There is an accumulation of gas in the alimentary tract, rapid decomposition, blood escapes from natural orifices, and blood stained froth from nostrils. Petechial haemorrhages may occur throughout the body, including the endocardium meninges and central nervous system. Superficial singeing of feet or muzzle, and internal or subcutaneous extravasation of blood in arboreal pattern may occur.

The condition is to be differentiated from anthrax, blackleg, hydrocyanic acid or nitrite poisonings (Blood and Henderson, 1963).

Heat Stroke, Heat Exhaustion, Heat Cramps and Dehydration Exhaustion

The heat regulatory mechanism is located in the hypothalamus.

The body temperature is increased in muscular activity, metabolism of

food, in disease, and in adverse environmental condition. Heat loss occurs from the body by respiration, mouth breathing, sweat, and salivation. Heat is dissipated by radiation and convection.

Some breeds of cattle can acclimatize to altered environmental conditions by their inherent capacity and also physical conditions, such as pigment of skin, hair coat, and subcutaneous fat.

The adrenals and thyroid play important roles in body heat regulation. The adrenal secretion exerts a calorogenic effect which is immediate and of short duration. The thyroid hormone has a long lasting but delayed effect in increasing the body temperature.

The body temperature is the balance struck between heat production and heat lost. The heat regulating mechanisms are depressed by anaesthetics, hypnosis and general bodily fatigue. During sleep the heat regulating mechanism is sluggish.

Heat Stroke

This is a condition resulting from hyperpyrexia, an accumulation of body heat. It can be brought on entirely with interference with heat radiation, but is hastened if there is an increase in heat production through exercise. The incidence is greatest during a prolonged heat wave.

High atmospheric conditions, moisture, poor circulation of air (less of oxygen), and exercise contribute to heat stroke. Fatigue, insufficient water, and salt intake play significant secondary roles. Overcrowding, forced exercise and inadequate ventilation precipitate heat stroke.

Death occurs due to the paralytic effect of extreme body temperature.

The body temperature rises from 107° to 115° F. The skin is dry. The patient is dull and depressed. Respirations become rapid with open mouthed breathing. The tongue is protruded and there is a frothy salivation. The conjunctiva is congested. Pupils are dilated but later contracted. The pulse is rapid and weak. There is palpitation of the heart. The patient sways and staggers, and goes down. Death occurs due to progressive paralysis of respiratory and vasomotor centers. The mortality rate is high.

Putrefaction rapidly sets in. The large veins are distended with partially clotted blood. The lungs and brain show intense congestion. The parenchymatous organs show degeneration (Covault, 1962).

Sun Stroke

Sun stroke is an acute form of heat stroke characterized by sudden onset, high temperature and high mortality.

Sun stroke occurs when the head is directly exposed to blazing sun for long periods. Exercise and overcrowding are not necessary in the production of sun stroke.

The direct rays of sun on the head produce intracranial dilation of blood vessels and hyperaemia of meninges and brain. Over heating of the brain causes paralysis and death following involvement of vital centers in the paralysis.

Symptoms develop suddenly. Restlessness, excitement, forced movements, and spasms of certain muscles occur. The temperature may be elevated and the pulse is weak. Death occurs with progressive paralysis of respiratory center.

Congestion of the meninges and brain with haemorrhages are seen. There is an accumulation of sanguineous fluid in the intermeningial space, and the brain may show oedema.

Heat Exhaustion

Heat exhaustion is a mild form of heat stroke and is characterized by gradual onset, depression, normal temperature and a low mortality.

The etiology of the condition is the same as that of heat stroke. High environmental temperature, prolonged severe muscular exercise, high humidity, and inadequate ventilation bring about the condition.

Upon sudden exposure to a high temperature, there is a dilation of the peripheral vessels, greatly increasing the vascular space. The heart rate and cardiac output increase and the blood pressure may fall a little. If additional physical work is performed the heart may not be able to maintain the blood pressure, and the condition known as heat exhaustion ensues. The skin is moist and sweating may be profuse. The pulse is rapid. Hyperpnoea and later dyspnoea indicative of cardiac insufficiency are observed. Body temperature may not be elevated. Weakness and muscular tremors may be followed by collapse.

The condition responds rapidly to rest and fluids by mouth.

Dehydration exhaustion is another form of heat exhaustion, where the patient is exposed to heat for a long period, without replacing the lost fluid.

In a hot environment with extensive sweating, the blood volume is reduced resulting in cardiac insufficiency and exhaustion to collapse. As

dehydration progresses, the heart rate rises and exertion becomes progressively more difficult. Exhaustion results, when the body has lost five percent by weight.

The patient suddenly collapses, unconscious with an extremely rapid heart rate. Rest and restoration of body fluids by mouth bring about rapid recovery.

Death from dehydration exhaustion (in humans) occurs suddenly, and may be preceded for sometime, by extensive oedema of legs, indicating the cardiac origin of the syndrome.

Heat Cramps

Heat cramps occurs due to deranged electrolyte balance, as a result of acute salt loss. This condition is seen more in horses which sweat profusely during hard work.

When the salt intake is less, and when the sodium chloride is lost by perspiration in large quantities, exertion will induce severe painful muscle contractures. After exertion these painful cramps will continue until the salt balance is restored. The administration of sodium chloride by mouth or parenterally will rapidly alleviate the cramps.

The animal does not sweat after onset of cramps (Best and Taylor, 1961).

Trauma

Among the extrinsic factors that produce neurological disorder of a noninfectious nature, trauma is one of importance.

This subject of trauma is generally dealt with under surgery. However the most common disorders of the nerve due to traumatic origin are being discussed briefly.

Traumatic injury to brain and spinal cord are not commonly met with in bovines.

The effects of trauma to the brain vary with the extent and severity of injury. Initially nervous shock occurs followed by death, recovery or persistent residual symptoms. Trauma is more common in parasitic diseases, such as cerebral nematodiasis, gid, filarid larvae, etcetera.

Trauma by external violence, produces haemorrhage and shock. A subdural haematoma may develop with a gradual onset of symptoms which vary with the site and extent of injury. A depressed fracture or haematoma produces pressure.

Trauma may result in concussion when the nerve cells get damaged without macroscopic changes. Concussion of the brain results by a violent blow on the head with or without a fracture of bones. The concussion causes a sudden displacement of the subarchnoid fluid in the brain, and the unconsciousness is produced due to local anaemia of the affected part, as a certain amount of blood is forced out of the capillaries into larger vessels.

The same sudden movement of intravascular blood, and the cerebrospinal fluid may cause numerous petechial haemorrhages into the subarchnoid spaces. The lesions are often on the side opposite to that of blow due to the impact of the brain on the opposite side.

With severe injury there is cerebral shock in which the animal falls unconscious with or without a transient convulsion. The animal may regain consciousness in a shorter or longer time.

During the unconscious stage, the pupils are dilated, respiration becomes slow and irregular. Bleeding from nose and ears may be noticed.

Blindness results if the optic cortex is affected. Hemiplegia occurs if the midbrain is damaged and traumatic epilepsy results when lesions are in the motor cortex.

Gross haemorrhages can be noted if the trauma is severe resulting in a bruise, and in concussion, haemorrhages can be seen only microscopically.

Diffuse compression of the brain and spinal cord result from pressure of extensive haemorrhage, tumors, abscess or oedema.

The symptoms start with irritability and convulsions, and later as the pressure increases, mental depression, somnolence, and coma set in. In a localized area of compression, softening and liquefactive necrosis develop if the patient lives for some time.

Pressure upon the cord leads to paralysis of the parts supplied from segments below the point of compression, starting with initial hyperirritability. It is of the upper motor neuron type that is spastic. Flaccid paralysis involves parts which happen to be innervated directly from the injured segment. Sensation ceases from the parts below the lesion.

The local pressure produces softening and liquefactive necrosis. Demyelination and loss of fibers in the motor tracts below, and sensory tracts above the injured segment occur.

Trauma to the spinal cord by way of dislocation of vertebrae or fracture of arch or body are not uncommon in bovines. The caudal vertebrae are often dislocated in the act of twisting the tail by human agency.

Complete, immediate, flaccid paralysis occurs behind the seat of injury because of spinal shock. This is followed by flaccid paralysis in the area

supplied by the injured segment and spastic paralysis caudal to it.

Fractures or dislocations in the spinal column result from falls or jumping or by external violence by man. Lesions consist of disruption of nervous tissue or its compression by displaced bone, or haematoma. Minor injuries result in concussion, or hyperaemia or oedema. In case only hyperaemia or oedema is present, the symptoms disappear as they resolve. If structural damage persists there is usually hyperesthesia in the area of lesion, flaccid paralysis in the same general area and spastic paralysis caudally. Traumatic lesions affect the whole cross section of the cord.

Spinal shock immediately after the injury, and flaccid paralysis sets in up and down the cord. A fall in blood pressure due to vasodilation, and profuse sweating sets in. Stretch reflexes and cutaneous reflexes disappear first, but return after some hours. Paralysis varies with the extent and severity of lesion. The extremities are affected and the animal will not be able to rise and lies down in sternal or lateral recumbency. There will be flaccid paralysis, disappearance of reflexes, and muscle wasting. If the lesion is located in cervical region all the four limbs get involved with spastic paralysis, without muscle wasting. Because of interference with the sacral autonomic nerve flow, paralysis of bladder and rectum may be evident.

Excessive mobility of spinal column, and malalignment of spinous process of vertebrae help in diagnosis.

If nervous tissue is not destroyed recovery may occur in two to three weeks. Recovery is improbable if the nervous tissue is destroyed.

Diseases of Peripheral Nerves

Many of the peripheral nerves which are superficially placed under the skin, in their courses may be damaged by trauma.

Damage to peripheral nerves by trauma, contusions, over stretching, or tumors may bring about paralysis of the muscles supplied by them. Radial paralysis, femoral paralysis in casting and securing the limbs, obturator nerve paralysis in distocies, facial paralysis in trauma, phrenugial paralysis in intoxication like botulism, vagal paralysis in case of lymphomas in mediastinal lymph nodes are some of the examples.

Allergic Encephalitis

Allergic encephalitis or post vaccinia encephalitis also can be classified under noninfectious neurological disorder due to extraneous factors.

When brain suspensions are injected parenterally, repeatedly or even a single dose is injected in an adjuvant to slow the absorption for longer period allergic encephalitis may develop. This condition is most commonly observed in humans or canines who are more frequently given antirabic treatment. (It is experimentally proved that a brain suspension without rabies killed fixed virus sets up the same condition.)

The symptoms commence with motor paralysis of one or more limbs which gradually extends to the whole body. Death is the usual outcome with a severe form of disease. The mechanism by which paralysis occurs has not been determined.

The lesions consist of destruction of myelinated tracts in the white matter. The lesions are large and promiscuous in the cerebellar peduncles and pyramids. Subcortical white matter is often affected and occasionally the corpus callosum is affected. The lesions appear, as irregular non-symmetrical areas of malacia, with destruction of myelin followed by usual glial and leucocytic response perivascular accumulation of lymphocytes in the region adjacent to malacia is observed. In the spinal cord, tracts in the myelinated white columns are similarly affected (Smith and Jones, 1961).

INTRINSIC FACTORS

Noninfectious neurological disorders due to intrinsic factors comprise (a) tumors and (b) symptomatic disorders.

Tumors

Tumors or neoplasia of nervous system are rare in the bovine species--few reports of their occurrence have been published so far.

Tumors in the brain or meninges, produce an increase in the intracranial pressure, and thereafter destroy the nervous tissue.

Symptoms develop slowly and are confined to the area supplied or related to the brain center wherein the tumor develops. The symptoms are aggravated with increased intracranial pressure produced by the tumor.

Clinical pathological examination would not help in the diagnosis of the condition. Fever is usually absent in brain tumors. Large tumors in the brain may not show definite signs in some cases, whereas small tumors may

produce pronounced lesions depending on the location of the tumor in the brain.

The tumors of the brain rarely metastasize. The differentiating features of benign and malignant tumors do not hold good in the case of brain tumors. Some tumors are extremely malignant in the sense of rapidity of growth and undifferentiated cellular characteristics, and the more so as they kill the host because of localization in some vital part and/or are inaccessible for surgical removal.

Neoplasms of the brain commonly arise from the neuroglia and all such tumors are termed gliomas. The gliomas have been classified as:

(1) Astrocytoma where the cells have round or elongated pale nuclei with granules of chromatin. These tumors arise in any part of the brain and are of less malignancy.

(2) Glioblastoma multiformae. The nuclei of these cells are pleomorphic in size, shape and position, deeply stained and more numerous. This type of neoplasm is more malignant and more common in man and is usually located in the cerebral hemispheres.

(3) Medulloblastoma, arises from embryonal glial tissue and consists of masses of round cells with deeply staining round nuclei. This resembles a malignant lymphoma and is located normally dorsal to the fourth ventricle, where it appears as a spherical discrete reddish gray mass.

(4) Ependymoma consist of cells of medium size with irregularly rounded or polyhedral nuclei centrally placed. The cells are arranged in a single zone of cells around a tiny open space and hence referred to as "rosette." These arise in the third or fourth ventricle.

(5) Oligodendroglioma consists of polyhedral cells separated into

compartments into trabeculae. The cytoplasm of the cells does not take the stain. It gives the appearance of small round nuclei, arranged along side of, but separated from the trabeculae (Smith and Jones, 1961).

The most common neoplasms that are reported in cattle are meningiomas, neurolemmomas, astrocytomas, lymphomatosis, and neurofibromas.

Meningiomas. Synonyms for this group are arachnoidal fibroblastoma and endothelioma.

These tumors are nodular, attached to duramater by a broad or narrow base. It indents or compresses the brain but never invades--plaquelike or diffuse patterns are seen.

They are densely packed, plump, polyhedral, well differentiated epitheloid cells set in vascular tissue stroma in whorled forms. Predominantly, fibrous, lipomatous, angiomatous and ossifying types occur.

"Probably most cases of spindle or round celled Sarcomas of meninges mentioned in earlier literature have been meningiomas" (Innes and Saunders, 1962).

Meningiomas were reported in two cows by Frankhauser (1947) as stated by Innes and Saunders (1962).

Meningiomas in cattle were reported by Tommasini and Mercato (1959). Meningioma and glioblastoma coexisted in a cow (Innes and Saunders, 1962).

Neurolemmomas (Schwannomas) or neurofibromatosis was reported (Moulux and Davis, 1953) in cattle affecting intercostals, brachial plexus, heart, and accoustic nerve.

Teternik et al., (1960) reported neuromas in hepatic plexus, intercostals, heart aorta and media stinum, liver, spleen and cervical plexus, muscles, lungs and lymph nodes in cattle.

Marigold (1958) described fatty nodules of a fibrous consistency--two to four centimeters in diameter occurring in the brachial plexus, and along the longitudinal sulcus of the heart. They were not pure neurinomas. They were identified to be neurofibromas.

Sullivan and Anderson (1958) reported schwannomas in the accoustic nerve in a six month old Hereford calf and in an 18 month old steer. These animals showed incoordination, and circling. The tumors were two inches by one inch by three-quarters inch in size. The essential tumor cells were elongated with fusiform nuclei and had a loose reticular structure in that of the calf and dense fibrous tissue in the steer.

Helve (1959) reported neurinoma in 142 cows slaughtered in Southern Finland from 1952 to 1956. A proportion of four in a 1000 animals had neurinomas. Neurinomas were not recorded in male stock.

Tumors of hypophysis cerebri (pituitary). Epiphysis cerebri (pineal gland) have not been reported in the bovine species. Cholesteoma or psammoma in the ventricles of brain, reported often in horse is not described to occur in the bovine species.

Melanomas often seen in the equine species, as a generalized form in the meninges of spinal cord are not noticed in the bovine species. Malignant lymphomatosis is seen affecting the sublumbar lymph nodes in cattle. Extension through the inter vertebral foramina leads to invasion of spinal canal. Neurologic symptoms are exhibited when the growing lymphoid mass compresses the spinal nerves, roots, ganglia and cord which undergo degeneration.

Squamous cell carcinoma of leptomeninges of brain in a cow has been reported by Peterson (1963).

Multiple neurofibrosis in a cow and calf and lymphosarcoma in another calf were reported in the nervous system by Simon and Brewer (1963). The two animals, the cow and the calf developed neurofibromata in identical location on the body (i.e.) left, mandibular area, tail head, submaxillary area and base of left horn.

The condition is suspected to be a heritable neurofibromatosis. In humans a heritable type of neurofibromatosis is known to occur under the name Von Recklinghausen's disease (Pack, et al., 1958).

Symptomatic Disorders

Symptomatic disorders of the nervous system due to impaired functions of other systems are noticed in cattle as well as in other species of animals. The symptoms exhibited are secondary and not primary.

Cerebral anoxia results secondarily to defects in circulatory system or respiratory system.

Anaemic anoxia results from excessive blood destruction as in piroplasmosis or Anaplasmosis.

Anoxic anoxia results due to improper ventilation in lungs--due to inflammation and/or oedema of lungs etcetra.

Histotoxic anaemia results when the tissues are severely damaged and unable to take up oxygen as in arsenic poisoning.

Stagnant anoxia results when there is arrest of circulation of blood in the tissues.

Cerebral disfunction occurs when anoxia is severe. Cerebral anoxia occurs in hydrocyanic acid poisoning, nitrate poisoning, terminal stages of

pneumonia and congestive heart failure.

Local anoxia of the brain occurs due to increased intracranial pressure by tumors, cysts, or cerebral oedema which diminish the vascular supply.

The central nervous system is extremely sensitive to anoxia and degeneration occurs if anoxia is prolonged for more than a few minutes.

Complete anoxia for 15 seconds is fatal.

When the onset of anoxia is sudden there is excitation followed by a period of loss of function. If recovery occurs a second period of excitation followed by return to normalcy of function takes place. When anoxia is less severe, there is dullness and lethargy and in severe cases of anoxia consciousness is lost.

Acute cerebral anoxia is manifested by paralysis of all brain functions, including flaccid paralysis and unconsciousness. Muscle tremor, beginning about the head, and spreading to the trunk and limbs, is followed by recumbency, clonic convulsions, and death.

In chronic cases, there is lethargy, dullness, ataxia, weakness, muscle tremors and convulsions.

Liver dystrophies including those caused by plant poisons are accompanied by nervous signs.

Severe mental depression occurs in *Crotalaria* poisoning. Many poisons cause neurological disturbances without producing any lesions in the brain such as organic phosphates, chlorinated, hydro-carbons, nicotine sulphate, carbon tetrachloride etcetra.

Many bacterial toxins including those of *Clostridium botulinum*, *Cl. tetani*, *Cl. perfringens* type D--produce neurological disturbances without any lesions. Lightning stroke and electrocution may cause death without

producing lesions.

Tick paralysis is another condition where nervous disturbances are noticed in young animals without demonstrable lesions in nervous system.

Nervous symptoms may be observed in other conditions, such as, hypocalcaemia, hypo-magnesemia, and acetonemia without producing any lesions in the nervous system.

NERVOUS DISORDERS IN BOVINES DUE TO NUTRITIONAL DEFICIENCIES

Vitamin A Deficiency

A deficiency of Vitamin A may be caused by an insufficient supply or defective absorption in the alimentary canal. In young animals Vitamin A deficiency symptoms are those of compression of brain and spinal cord.

In adult animals, night blindness, corneal keratinization, infertility, defective hooves, and loss of weight are the signs of Vitamin A deficiency.

Congenital defects are common in the offspring of deficient dams.

Moore et al. (1935) reported constriction of optic nerve at the entrance of optic foramin and consequent blindness in calves kept on Vitamin "A" deficient diet.

Deficiency of Vitamin A retards endochondral growth at a time, when bone is being substituted for cartilage, thereby interfering with normal bone growth. This interference causes long bones to become thicker and shorter resulting in disproportionate bone formation around the central nervous system (Blackmore et al., 1957). It was reported by Mellanby (1933, 1939, 1944) that Vitamin A deficiency caused arrest of epiphyseal growth,

whereas appositional growth on the facial bones continued. Pressure was thus exerted on the nervous tissue. Overcrowding of the spinal canal and cranial cavity, resulted in production of multiple herniations of brain, and a dislocation of it towards the foramin magnum. Construction of optic foramin results in blindness due to changes in optic nerve and abnormalities in retina.

Blackmore et al. (1957) observed arrest of growth, watering of the eyes, papilloedema, oedema of the retina, excitation such as losing control of limbs and lying with legs and neck extended, flaccid muscles, doming of skull in calves with Vitamin A deficiency. Convexity and thickening of parietal and frontal bones, "packed in appearance" of the brain were prominent. Confirmation of various parts of brain was altered, with the medulla, pons and cerebral peduncles forming a more upward curve than usual, and an exaggerated downward direction of cerebellum and olfactory areas. Arching of the mid-brain area was associated with bony changes on the base of the skull. The cerebrum and cerebellum showed compression changes in the dorso-ventral direction. The state of ventricles and aqueduct of Sylvius suggested an increase of cerebrospinal fluid. The optic tracts and chiasma were subjected to pressure between the brain and bone. The optic nerve was elongated and assumed an S-shaped curve consistently. In some cases the optic nerve was surrounded by a dense mass of fibrous tissue, with adhesion to bone. The optic nerve was surrounded by a pigmented sheath derived from duramater (i.e.) an extension of the meningeal sheath. This covering contained many small blood vessels and nerve fibers. There is a proliferation of the neurolemmal sheath into the optic nerve. The nerve became obliterated at the optic foramin and is divided into two segments, covered by a sheath of neurolemma

and joined together by a loose connective tissue.

Cysts were noticed in pars anterior and intermedia of the pituitary without changes of pressure atrophy.

It can be concluded from the experimental results that fibrosis, lengthening, constriction of the optic nerve at the optic foramen with increased intracranial pressure, doming of head and nervous symptoms are the symptoms of Vitamin A deficiency in calves.

Symptoms of hydrocephalus due to increase intracranial pressure, and oedema of brain, has been discussed under congenital and hereditary disorders.

Thiamin Deficiency

Thiamin is synthesized in mature cattle by the rumen microflora, for young nursing calves oral thiamin is necessary. Thiamin deficiency in calves produces ataxia, tremor, convulsions (Innes and Saunders, 1962).

Biotin. A deficiency of biotin produces a paralysis of the hindquarters (Wise et al., 1946).

Generally speaking Vitamin B complex deficiencies do not occur in cattle, since they are synthesized in the rumen in cattle.

Keslar et al. (1950) stated that even a two week old calf can synthesize many of these compounds in rather large quantities.

Mineral Deficiencies

Certain minerals such as calcium and magnesium produce nervous signs in the bovine species when deficient in nutrition, but lesions diagnostic of the

deficiency diseases have not been described.

Calcium. The metabolism of calcium is controlled by the parathyroids. The parathyroid controls withdrawal of calcium from bone to maintain the calcium level of blood when calcium is deficient in the diet.

Parathyroid removal results in hypocalcaemia.

Hyperirritability of the peripheral nerves is the main symptom observed in hypocalcaemia. The blood calcium level in cattle is seven mg. of calcium per 100 ml. of blood. A fall in the calcium blood level (hypocalcaemia) reflects as a nervous disorder.

Symptoms of hypocalcaemia are observed when there is inadequate supply of calcium in feed or in disease of the parathyroid, liver disfunction, or in disease of kidney and in milk fever. Interference with the normal flow of bile, coupled with a diet rich in fats may produce steatorrhea and formation of calcium soaps in the intestine to the point that hypocalcaemia results. Likewise in kidney disorders (nephrosis), sufficiently severe due to an inability to excrete phosphates in the urine, the excretion of calcium phosphate into the alimentary tract deprives the blood of its proper level of calcium.

Hypocalcaemia depresses the nervous system and general body activity in milk fever contrary to what occurs in parathyroid tetany.

Milk fever (referred to as parturient paresis or parturient apoplexy) occurs when there is a sudden heavy excretion of milk which is rich in calcium. Due to sudden and heavy loss of calcium, the hormonal mechanism is thrown out of its normalcy and this hormonal imbalance exhibits itself as hypocalcaemia. In milk fever, there is a primary irritation of the nervous system, with symptoms of excitement and muscular incoordination. This state

is very soon followed by nervous depression, exhibited by the somnolence and recumbency of the animal however, no lesions in the nervous system can be detected.

Magnesium. Bovine blood contains two mg. of magnesium in 100 ml. of blood.

There is some evidence indicating that biochemically the essential role of magnesium is in the transmission of impulses at the neuromuscular function; a low concentration of magnesium, and a low ratio of magnesium to calcium in the surrounding tissue fluids, facilitating perhaps unduly, the formation of acetylcholine which is presumably essential feature of the transmission of impulses (Moore et al., 1938).

A fall in the magnesium level below 0.7 mg. per 100 ml. in blood, causes nervous hyperirritability and a great excess causes nervous depression, unconsciousness, and death.

In experimentally deficient calves symptoms described are opisthotonus, very frequent movement of fixed depression of the ears, greatly exaggerated scratching, kicking at the belly, or twitching of the skin in response to slight stimuli, spastic extreme flexion of the carpus in walking, salivation, exophthalmos and apprehension occur. These signs are intermittent and initiated by excitement or exercise. After days or weeks, terminal tonic and clonic convulsions lasting one or two hours are fatal.

Necropsy lesions were limited to agonal haemorrhages, heart, intestinal and mesenteric serosa and congestion of viscera. Neither the central nervous tissue nor the muscles showed anything of significance (Blaxter et al., 1954).

A similar condition occurs in adult cattle, with identical symptoms and lesions, occurring naturally, when cattle are pastured for some time exclusively on lush and succulent grasses. The condition is referred to as

"grass tetany," "grass staggers," or "wheat poisoning."

The blood of the affected animals shows hypomagnesemia and some hypocalcaemia, even though the grasses on analysis never revealed any deficiency of both.

Some calves mainly dependent on milk, suddenly develop tonic clonic convulsions and die in an hour or so. Investigations indicated, hypomagnesemic condition in these calves (Blaxter et al., 1954). Necropsy reveals no lesions. Udall (1947) described a similar condition known as winter tetany in cattle.

The blood level of magnesium in winter falls to half the normal level for reasons not known. When exposed to winter conditions on pasture, some cattle abruptly develop tetanic symptoms, which are fatal in many instances.

Copper Deficiency. Copper is present in all tissues including the central nervous system in the animal body.

A condition known as "sway back" or "enzootic ataxia" in sheep and cattle has been described in certain areas where copper is deficient in the soils. Incoordination and weakness of the hind limbs, spastic paralysis, and sometimes blindness are the reported symptoms in affected animals.

Newborn lambs are severely affected. The lesions consist of demyelination, and even extensive softening, necrosis, and disappearance of much of the white matter in central portions of cerebral hemispheres. In severe cases there is little left of the hemisphere but the cortical shell of gray matter. In less severe cases, areas of demyelination, symmetrically located with "gitter" cells, and necrotic nerve cells occurs. There is no inflammatory reaction. Sheep are more frequently affected than cattle.

Cobalt deficiency produces a condition called "enzootic marasmus." It

is a cachetic syndrome in which cattle and sheep, after grazing on certain pastures for months, develop anorexia and wasting followed by death.

In sheep the condition in Australia is called "coast disease." Both copper and cobalt were found to be lacking and the lesions observed are demyelination and destruction of certain tracts in the spinal cord.

NONINFECTIOUS NEUROLOGICAL DISORDERS DUE TO POISONOUS PLANTS AND CHEMICALS

Poisons of plant or chemical origin, when in sufficient concentrations produce their evil effects in different ways. Some poisons which are severe irritants kill the tissues with which they are in contact. When ingested, they produce severe inflammation and necrosis of the alimentary tract. Strong chemical acids and latex of plants like *Euphorbia* species fall under this group.

Some poisons have little or no immediate effect, but after entering the circulation, they attack the highly specialized parenchymatous cells of the liver and kidney. Weeds come under this category. *Senecio jacobaea*, *Lantana camara*, *Heliotropium europaeum*, *Tribulusterrestris* are some of such plants. These plants contain hepatotoxic principles and so damage the liver. Secondary nervous symptoms such as derangement of brain may also occur.

Some poisons interfere with the vital functions of nervous system, without producing any pathological changes. Organic phosphates and Strychnine are some examples of this group. Some poisons produce petechial or ecchymotic haemorrhages, while some haemolyse the erythrocytes. Some destroy the bone marrow and some block vital enzyme systems. Some poisons act in more than

one way.

The poisonous plants are of many families. The poisonous plants are different in different countries. Their geographical distribution is dependent on the climate of the country. Plants listed to be poisonous in one country may not be existant in other countries.

The active principles of many toxic plants have not been identified and experimental study of their poisonous effect on animal body has not been carried out. Many details about some known poisonous plants are obscure.

Since the subject under discussion relates to neurological disorders, poisonous plants that cause nervous malfunctions alone will be dealt with.

Strychnine

Strychnine is the active principle of *Strychnos nuxvomica*. It is a native of tropics and subtropics. The seeds are flattened and circular. The active principle is contained in the seed.

Symptoms of strychnine poisoning are intermittent tonic spasms initiated by noise or external stimuli.

Spasms occur due to hyperirritability and lack of normal inhibitory restraint in the spinal part of the spinal reflex area. It is a cumulative poison if taken in small doses for prolonged periods as a nervine stimulant.

There are no post-mortem changes except probably petechial resulting from the anoxia, incident to stoppage of respiration during the spasms (Cox, 1957).

Loco Poisoning

Loco poisoning occurs when the plant Astragalus earlei, or A. mollissimus is fed to animals in very large quantities over prolonged periods of at least a couple of months.

The active principle is locoine. Sensory motor derangement, ataxia of the limbs, ascending paralysis, defective vision are the chief symptoms.

Lesions are only microscopic--degeneration and necrosis of neurons in brain constitute the chief lesion (Nokolds, 1896 and Schwartzkopf, 1898).

Vetchlike Astragali

These are small leguminous plants in the Rocky Mountain ranges. They are known as timber milk vetch and include Astragalus decumbens, A. cenvallarius, A. hylophilus and A. compestris.

Only ruminants are affected. Symptoms are incoordination of the hind limbs and nervous weakness, sinking of the hind quarters and knuckling of the fetlocks. The metatarsal and phalangeal joints are abnormally relaxed and poorly controlled. No disturbance of sensation is noticed.

Acute hypertrophy of the heart is a constant finding.

Lesions in central nervous system have not been described (Smith and Jones, 1961).

Claviceps paspali

This ergot is smaller than Claviceps purpurea--and grows upon Dallis

grass.

Animals develop symptoms after ingesting parentized seed heads of grass.

Symptoms are nervous in character and manifest themselves in nervous hyperirritability and excitability.

Belladonna (Deadly Night Shade)

The foliage and unripe berry of Atropa belladonna contain the active principle--atropine.

Symptoms are loss of nervous control, incoordination followed by convulsions and death usually within 12 hours after ingestion. Mydriasis, falling of temperature and failing of heart are typical symptoms.

Lesions in the central nervous system are not noticed (Smith and Jones, 1961).

Hemlocks

The plant Conium maculatum (hemlocks) contains an active toxic principle conium which acts as nervous depressant. Symptoms of poisoning include loss of muscular strength, gradual loss of power of locomotion, generalized tremors, followed by coma and death in one to two hours.

In cattle, lacrimation, salivation, dyspnoea and bloody diarrhoeas have been described in one to two hours following ingestion of the plant; and death occurring in one to two days.

The active principle conium is a slow cardiac depressant; as a consequence of slow cardiac failure, there is widespread passive congestion,

most noticeable in the lungs, liver, and the coronary vessels. In cattle, localized catarrhal enteritis is frequently noticed.

Cicuta douglasii also known as water hemlock, growing in wet areas in the Rocky Mountains in the United States, when ingested, produces severe convulsions and death (Durrel et al., and Aggio, 1907).

Lathyrus Poisoning

Lathyrus sativa (leguminaceae) is grown in several countries. Seeds (peas) are edible and are used as human food.

Poisoning only occurs when considerable amounts of plants are eaten over a long period of weeks or even months.

Posterior paralysis is the prominent symptom in animals and man. There is degeneration of the neurons in the spinal cord, accompanied by gliosis, and ultimately atrophy of the cord. The lesions are suggested to be of an inflammatory and not degenerative nature. In cattle, blindness, torticollis, and anesthesia are additional symptoms. Death occurs by respiratory failure.

In addition to the lesions in the spinal cord, there are mild chronic enteritis, terminal subepicardial haemorrhages (asphyxiative) and pulmonary congestion (Smith and Jones, 1961).

Larkspur poisoning. The plant Delphinium causes poisoning in cattle and horses on the ranges of the western United States. Symptoms appear in a short time after the plant is eaten, and terminates favorably or unfavorably within 24 hours. Symptoms include salivation, repeated swallowing, neuromuscular weakness, incoordination, staggering gait, muscular quivering and convulsions followed by death.

The post-mortem lesions are acute catarrhal gastroenteritis and widespread venous congestion typical of gradual cardiac failure (Marsh et al., 1916).

Phalaris tuberosa. A native plant of Australia and New Zealand produces the condition called "phalaris staggers" (McDonald, 1942) in sheep and cattle. This perennial grass, in certain copper deficient areas when grazed by sheep and cattle, produces symptoms of incoordination, muscular tremors, excessive salivation, and inability to swallow. When the animal tries to run, it falls to the ground and struggles vigorously in futile attempts to rise. This may be preceded by violent behavior and convulsions. Symptoms were similar in sheep and cattle.

Post-mortem lesions consist of degeneration of the nerve fibers in the lateroventral tracts in the zone surrounding gray matter. The lesions were symmetrical and extended throughout the entire length of the cord. This condition phalaris staggers has not been reported in the United States.

Some plants contain hepatotoxic substances and cause a syndrome of liver insufficiency, photosensitization and marked signs of central nervous system derangement. The following plants are the examples:

Ragwort -- Senecio jacobaea.

Tarweed -- Amsinckia intermedia.

Puncture vine -- Tribulus terrestris.

Lantana -- Lantana canara.

Heliotrope -- Heliotropum europaeum (Blood and Henderson, 1961).

Botulism is a rapidly fatal, motor paralysis caused by the ingestion of the toxin of Clostridium botulism, which organism proliferates in decomposing animal or vegetable matter.

It occurs in cattle suffering from protein or phosphorus deficiency on range. The disease is invariably fatal.

The causative organism is the spore forming anaerobe Clostridium botulinum which proliferates only in decaying animal or vegetable matter. It elaborates a stable and highly lethal toxin. Ingestion of the preformed toxin causes the disease. Cattle are affected with types C. and D. of Cl. botulinum.

Outbreaks occur in summer or drought conditions when the feed is sparse. The source of infection for cattle is always a carrion. When cattle subsist on a phosphorus deficient diet, and manifest osteophagia and the ingestion of carrion, the disease is likely to occur in an outbreak form. Spoiled silage may be a source of botulism toxin.

The toxins are neurotoxins and produce functional paralysis, without the development of histological lesions. When toxins are injected parenterally, much smaller doses are required to cause death than when it is ingested. The site at which the neuromuscular transmission is impeded is probably at the synapses of efferent parasympathetic and somatic motor nerves, where there is interference with the secretion of acetylcholine, the chemical mediator of nerve impulse transmission (Wright, 1955). A true flaccid paralysis develops and the animal dies of respiratory failure.

The symptoms develop three to seven days after the animal ingests the toxic materials. There is progressive muscular paralysis affecting particularly the limb muscles and the muscles of the jaw and throat. Paralysis commences in the hind quarters and progresses to the fore quarters, the head and the neck. Skin sensation is retained. The tongue is paralyzed and hangs from the mouth and the animal is unable to chew or swallow.

Constipation may be present. Paralysis of the chest muscles results in a terminal abdominal type of respiration. Sensation and consciousness are retained until the end. The course is afebrile throughout.

Snake Bites

There are two types of venomous snakes--the adder type and the cobra type.

The adder type, including the pit viper, copperheads and rattlesnakes produce two toxins--a neurotoxin and a haemotoxin. The bites by this type cause severe local swelling at the site of the bite. The bite of the cobra type including cobras and coral snakes may produce no obvious local lesion, the venom containing chiefly a neurotoxin.

The toxins include necrotizing and coagulant fractions, as well as neurotoxic and haemolytic fractions.

The neurotoxins cause initial stimulation of the central nervous system followed by paralysis. (Effects of the other toxins include local tissue necrosis, capillary damage and necrosis.) (Gordon, 1958).

Tick Paralysis

A toxin is secreted by adult female ticks--Dermanysis andersoni. The toxin interferes with liberation or synthesis of acetylcholine at the motor end plates of the muscle fibers (Emman and McLenan, 1959).

There is an ascending, flaccid paralysis commencing with incoordination of the hind legs followed by paralysis of the fore limbs and chest muscles.

Pupils are widely dilated and death is due to a respiratory paralysis.

Young calves are more susceptible than adults.

Lead Poisoning

Calves are frequently affected with lead poisoning by licking of painted surfaces. Orchard sprays contain lead arsenate is another source. Most of the lead that is ingested in any form gets excreted in faeces in the form of insoluble lead complexes.

In acute lead poisoning, it is deposited in the liver and renal cortex and in chronic poisoning in the bones. The metal is eliminated through bile and urine.

Young animals (calves) are more susceptible than adults. A lethal dose is 0.25 mg. lead per kg. body weight.

The toxic effects of lead are manifested in three ways, encephalopathy, gastroenteritis, and degeneration of peripheral nerves. In general, acute nervous system involvement occurs following ingestion of large doses in susceptible animals, such as calves; alimentary tract irritation following moderate doses. The mechanism by which the nervous signs of encephalopathy and the lesions of peripheral nerve degeneration, are produced is unknown although there is some increase in cerebrospinal fluid pressure.

Constipation is generally present. Acute symptoms are noticed in calves and subacute symptoms in adults. The course is 12 to 24 hours in calves. Affected calves commence to stagger and show muscle tremor particularly of the head and neck with champing of jaws and frothing at mouth. The animal collapses and intermittent tonic clonic convulsions develop and may continue

until death. There is hyperaesthesia to touch and sound and the pulse and respiratory rates are increased. In adults in some cases, blindness, mania, charging into fences, attempting to climb walls, and pressing of the head against objects are manifested. The gait is stiff and jerky and the progress is impeded. Death usually occurs during a convulsion and is due to respiratory failure. The adult animal lives for four to five days.

In acute cases there are no gross lesions at necropsy. In long standing cases, there may be a gastroenteritis, diffuse congestion of the lungs and degeneration of liver and kidney. Epicardial haemorrhages are common. Haemorrhages may be present in meninges. There is increase of cerebrospinal fluid.

There is myelin degeneration and eventual necrosis of axons of motor nerves.

Mercury Poisoning

Mercurial poisoning occurs accidentally in bovines. Biniodide of mercury used as a counter irritant may accidentally be licked by the patient.

Fungicides are also a source of mercurialism. Coagulative necrosis of gastric mucosa, destruction of uriniferous tubules, and ulcerative colitis are the chief lesions. Diarrhoea, anuria, and uraemia are the symptoms.

Swine are the most susceptible species for mercury poisoning and symptoms in swine are entirely related to nervous system. Pigs become blind, with ocular lesions, become weak, and incoordinated. Besides the lesions described in cattle, swine show cloudy swelling of the salivary glands, pancreas and liver. The nervous system is most severely affected in pigs.

Throughout the brain there is neuronal degeneration and necrosis. Demyelination of nerve tracts extends into the cord, and occasionally encephalomalacia and myelomalacia are seen. Peripheral nerves also show pronounced demyelination. Often many peripheral nerve fibers are reduced to hollow cylinders.

These nerve lesions do not occur in cattle.

Chlorinated Hydrocarbon Poisoning

Insecticides such as D.D.T., chlordane, lindane, toxophene, strobane, aldrin, dieldrin, etc. are all chlorinated hydrocarbons.

Accidental poisoning occurs while dipping or spraying the cattle and while deticking or treating skin diseases.

Symptoms of poisoning occur within minutes or hours. Spasmodic twitching and quivering of various groups of muscles including those of the eyelids are the first symptoms observed. Incoordination and convulsions soon occur. Body temperature rises to approximately 115° F. due to muscular activity and derangement of the heat regulating center. Dyspnoea and cyanosis are observed.

Lesions include petechiae and ecchymoses on and in the heart, and congestion and oedema of lungs. Some have reported Nissl's degeneration, and necrosis of neurons, especially in the ganglia of medulla and the brain stem (Blaxter, 1959).

Some have found no central nervous system lesions except congestion and increase in cerebrospinal fluid (Phillip et al., 1951). In delayed cases, acute toxic hepatitis, and acute tubular nephritis have been reported.

The nervous symptoms noticed may be due to increased cerebrospinal fluid

or due to derangement of liver or both.

Organic Phosphate Poisoning

Many insecticides are now on the market which are organic phosphates under different trade names--as TEPP, Parathion, Malathion, Diazinon, Chlorthion, Dipterex, Ruelene, etcetra. These are highly poisonous compounds and accidental poisoning may occur in cattle. Their toxic effect is purely functional, and no residual defect persists in recovered animals.

Spraying of these insecticides in the orchards and accidental contamination of the pasture are some hazards.

Organic phosphates are now employed as anthelmintics and systemic insecticides, such as CoRal, Neguvon, Coroxon, Korlon, Ronnel, Trolene, Trichlorphon, etcetera. Indiscriminate treatment with these products leads to poisoning.

The inactivation of cholinesterase by the organic phosphates, cause an increase in acetylcholine in tissues, and increased activity of the parasympathetic nervous system (McGirr et al., 1953). Over stimulation of the parasympathetic system produces tremors, salivation, pain, vomiting, diarrhoea, sweating, and increased bronchial secretion. Weakness of voluntary muscles indicate excess of acetylcholine (since the enzyme that hydrolyses acetylcholine is inhibited) in the myoneural junction. Death occurs due to respiratory failure.

Chronic experimental poisoning produced serious structural changes in the peripheral nerves and tracts of the spinal cord. The irreversible effects on nervous tissue are not clearly understood. These poisons have a

cumulative effect due to the increasing inactivation of the enzyme cholinestrase (Jolly, 1957). Post-mortem lesions are minor. Haemorrhages appear in the heart, lungs, and gastrointestinal tube. Pulmonary congestion and oedema are present.

NONINFECTIOUS NEUROLOGICAL DISORDERS OF UNKNOWN ETIOLOGY IN BOVINE

Polio-encephalomalacia of Cattle

This is a noninfectious disease of cattle and sheep reported from different areas and is of an unknown etiology. Several reports are published in the recent literature and the symptoms and lesions described in the syndrome are similar. The disease polio-encephalomalacia was reported in cattle and sheep by Jensen et al. (1956), and Terlecki et al. (1961); in sheep only by Innes et al. (1955), Hartley et al. (1959); and only in cattle by Neal et al. (1960), Elliot (1958), and Howel (1961). The disease occurred sporadically in cattle and sheep. It is characterized by a syndrome indicative of increased intracranial pressure and at necropsy by cerebral oedema and symmetrical necrosis of the cerebral cortex. The disease is reported in North America, New Zealand and in the United Kingdom. Morbidity is 25 percent in cattle and sheep and mortality rate is 90 percent for feed lot cattle, and 50 percent for cattle at pasture.

The disease is not transmissible--change of feed or pasture often resulted in termination of outbreak.

Reports of prevalence of disease in pigs has been reported by Dove (1957).

Other etiological agents suspected are (1) enterotoxins produced by Cl. perfringens type D. and (2) organic mercury compounds.

The cerebral oedema produced in pigs is similar to the one seen in salt poisoning.

Since the causative agent could not be ascertained, the disease is classified as a disease of unknown etiology.

The disease occurs in cattle under two years of age (generally 12 months to 18 months of age). It is also termed forage poisoning and blind staggers in Colorado.

In acute cases in cattle there is sudden onset of blindness, muscular tremor, particularly of the head, salivation and opisthotonus and in some animals convulsions. No lesions could be observed in the eyes. Visible mucous membranes become injected, respiration and pulse rate are accelerated, and the temperature in some cases elevated (generally normal). The animal exhibits masticatory movement, and profuse salivation, with occasional twitching of facial and ear muscles as the disease progresses. Initially there were some intervals of normality, but subsequently the syndrome is constant. Death occurs in one to two days preceded by coma.

In less severe cases, the animals may show head pressing but do not go down. Recovered animals are blind or stupid. Lesions are limited to the central nervous system. The changes in cerebral cortex are promiscuous. There is focal, later diffuse liquefactive necrosis destroying most of the gray matter. The white matter was normal. Cerebral oedema with compression and yellow discoloration of dorsal cortical gyri is evident, and the cerebellum is pushed back into the foramen magnum with distortion of its posterior aspect. In recovered animals there is macroscopic decortication

about the motor area, and over the occipital lobes.

Histological lesions are restricted only to the gray matter, and are necrotic in type.

In the cerebellum, necrosis of the granular layer preceded by loss of purkinje cells, and occasional cystic cavities were observed, but in most cases debris was removed promptly by gitter cells.

The disease may be confused by its clinical symptoms with acute lead poisoning, enterotoxaemia, mercury and arsenic poisonings, and hypovitaminosis A., which also occurs in calves. The history assay of faeces, urine and blood help in differentiating the conditions.

Infectious diseases are febrile, unlike polio-encephalomalacia.

Arthrogryposis and Hydranencephaly in New Born Calves

This is another disease of unknown etiology.

The two conditions arthrogryposis (curly calf) showing permanent joint contracture, and hydranencephaly (a compensatory replacement of fluid of missing cerebral cortical tissue in contradistinction to atrophy of brain tissue caused by fluid pressure as in hydracephalus) are considered to be different manifestations of the same disease.

Both of these conditions appeared as an outbreak in the same herd at the same time. The two conditions appear to be varying degrees of the one disease, and their variations may reflect the age at which damage to the developing nervous system occurred.

Calves with uncomplicated hydrancephaly are born normally but are blind and imbecile; those with arthrogryposis frequently cause foetal dystocia

because of severe contracture, which may be inflexion or extension and affect one or more limbs. Wryneck may also be present and there is severe muscle atrophy. At necropsy the cerebral hemispheres are entirely replaced by fluid in the calves with hydrancephaly. In the calves with arthrogryposis, there is a severe muscle degeneration and fixation of the joints by contracted tendons without abnormality of the joint surfaces.

The cause of the disease is suspected to be one of the following: (1) infection, (2) mineral deficiency, or (3) plant poison with *Jussieu repens* (Blood, 1956).

Neuronopathy and Pseudolipidiosis in Aberdeen Angus Calves

Whitten and Walker (1957) have reported this condition in Aberdeen Angus calves in Australia.

The disease is clinically characterized by ataxia, intention tremor (a tremor which is intensified by voluntary movement, and may cease when the animal is at rest) and failure to grow.

Signs appeared at one to 15 months of age and the disease progresses over a period of three to four months. The first sign is the swaying of the hind quarters especially after exercise or excitement. A fine head tremor develops and is accompanied by aggressiveness, and a tendency to attack. Ataxia increases, and the movements of the limbs are jerky, incoordinated, exaggerated and misplaced and the animal falls easily. Terminally ataxia is very severe or paralysis may set in.

There are no constant macroscopic findings at necropsy. Histologically there is gross abnormality of nerve cells throughout the brain, without

apparent damage to axons. Vacuulations of the larger nerve cell bodies is a characteristic finding. Similar lesions are present in the reticulo-endothelial cells of the lymph nodes. The cause of the disease is unknown. It is suggested to be an inherited factor.

SUMMARY

The noninfectious neurological disorders of bovines have been classified on the basis of their etiological significance and discussed.

Noninfectious neurological disorders are due to

- (1) Congenital and/or hereditary defects,
- (2) Environmental and extraneous factors,
- (3) Intrinsic factors,
- (4) Nutritional deficiencies,
- (5) Toxic plants and chemicals, and
- (6) Unknown etiology.

Congenital defects are those which originated in early embryonic life, and hereditary defects are those of genetic structure.

Most of the congenital defects reported have been traced to be hereditary defects also.

Failure of development of the neural groove anteriorly, results in congenital deformities, such as hydrocephalus, bulldog calves, and cleft palate; while failure of development of the posterior pole of the neural groove results in defective hind legs. Failure of the neural groove to close in the embryonic life gives rise to meningocele, spina bifida, etcetra.

Most of the defects which are described as hereditary are due to

recessive characters. These defects are not sex linked. The hereditary characters manifested as cerebellar hypoplasia, agenesis, congenital ataxia, spastic paresis, and congenital posterior paralysis in newborn calves are recessive traits. These undesirable characters are expressed only when both the parents carry the recessive characters (double recessive). This is the reason why most of the defects are consistently observed in line breeding and inbreeding operations.

These defects can be eliminated by studying the heredity of the bull and avoiding line breeding and inbreeding.

Nervous disorders due to environmental factors such as lightning, heat stroke, sun stroke, and heat cramps have been discussed comprehensively. Other extraneous factors, which produce nervous disorders, such as trauma and allergy have been described in detail covering the etiology, symptomatology and histopathological changes.

Tumors affecting the nervous system and symptomatic disorders of nervous system, which are secondary to other systemic disturbances have been classified under intrinsic factors, and the correlation of symptoms to lesions have been explained. The various types of neoplasms affecting the nervous system, have been enumerated and their structures described.

The deficiencies of Vitamin A, thiamin and biotin, and mineral deficiencies of calcium, magnesium, copper and cobalt manifest in nervous disorders. The lesions and symptoms of these deficiency diseases have been described.

Action of toxic principles of plant or chemical origin on nervous tissue, and the reaction of nervous tissue, as exhibited by lesions have been discussed.

The list of poisonous plants described in this study relate only to

those of the United States. Many more plants are to be identified, and the nature of action of the toxic principles of the poisonous plants, have to be investigated on an experimental basis.

The lesions produced in nervous system in chemical poisoning--such as lead, mercury, chlorinated hydrocarbons and organic phosphates have been described, and the clinical signs of toxicity have been correlated with lesions produced.

The toxin of Ci. botulinus has been dealt with under noninfectious diseases, since the condition can be construed as an intoxication.

Snake venom contains a neurotoxic principle and is therefore covered under toxicology.

The etiology of certain nervous diseases such as polio-encephalomalacia is often speculative. Polio-encephalomalacia, as reported from the literature, cannot be experimentally produced and no experimental proof supports any of the theories concerning its etiology.

Generally speaking, noninfectious neurological disorders in bovines are very few. The hereditary defects can be eliminated by avoiding inbreeding and line breeding.

Nervous disorders due to extraneous and environmental factors can be minimized by proper management and human care.

Nutritional deficiencies can be compensated by providing adequate vitamins and minerals. Toxic plants in pastures can be weeded out or such pastures can be avoided.

The accidental poisoning by lead and mercury are less common than that of chlorinated hydrocarbons and organic phosphates.

The recent additions of organic phosphates as insecticides are serious

hazards to livestock when used indiscriminately. Utmost care is to be taken when these insecticides are used.

The diseases of unknown etiology are to be investigated.

ACKNOWLEDGMENTS

The writer wishes to acknowledge with grateful thanks, the valuable advice and instructional guidance given by Dr. J. L. Noordsy of the Department of Surgery and Medicine.

Sincere appreciation is extended to Dr. J. E. Mosier, Professor of Surgery and Medicine, Kansas State University, for the suggestions offered in preparations of this report.

The writer is very much thankful to Dr. J. L. Noordsy, Dr. J. E. Mosier and Dr. M. C. Mussman, whose human sympathy and interest in the writer, has been an inspiration and encouragement.

Thanks are also extended to other members of the teaching staff, who have been helpful in instruction during the year.

REFERENCES

- Aggio, C. (1907). Two interesting outbreaks of vegetable poisoning. *Amer. Vet. Rev.*, 32:3680-3690.
- Anderson, W. A., and C. L. Davis (1950). Congenital hypoplasia in holstein-freisian calf. *Jour. Amer. Vet. Med. Assoc.*, 107:460-461.
- Baker, M. L., L. C. Payne, and G. N. Baker (1961). The inheritance of hydrocephalus in cattle. *J. Hered.*, 52:135-138.
- Becker, R. B., C. J. Wilcox, and W. R. Pritchard (1961). Spastic syndrome in cattle. *Jour. Dai. Sci.*, 44:542-544.
- Berger, J., and J. R. M. Innes (1948). Bulldog calves (Achondroplasia or Chondrodystrophy) in a freisian herd. *Vet. Rec.*, 60:57-58.
- Best, C. H., and N. B. Taylor. The physiological basis of medical practice, 1961 edition, pp. 893-894.
- Blackmore, C. F., C. W. Ottoway, K. C. Sellar, E. Eden, and T. Moore (1957). The effect of diet deficiency of vitamin A on the development of skull, optic nerve and brain. *Jour. Comp. Path.*, 67:277.
- Blackwell, R. L., J. H. Knox, and E. E. Cobb (1959). A hydrocephalic lethal in hereford cattle. *Jour. Hered.*, 50:143-148.
- Blaxter, K. L., and J. A. F. Rook (1954). The magnesium requirements of calves. *Jour. Comp. Path. and Therapeutics*, 64:176-186.
- Blaxter, J. T. (1959). Some observations on histopathology of Aldrin poisoning in lambs, *J. Comp. Path.*, 69:185-191.
- Blood, D. C. (1956). Arthrogryposis and hydranencephaly in new born calves. *Aust. Vet. Jour.*, 32:125-131.
- Blood, D. C., and T. H. Henderson (1963). A textbook of veterinary medicine. Baltimore: Williams and Walkins Co.
- Calist, V., and Chichivi (1961). Amyotrophic paresis of forelegs in China cattle. *Alta. Soc. Ital. Sci. Vet.*, 15:300-305. Abstracted from *Vet. Bull.*, para. 4318, 1962.
- Coles, C. L., and L. A. Moore (1942). Hydrocephalus--A lethal in cattle. *Jour. Agri. Res.*, 65:483-491.
- Covault, C. H. (1962). Diseases of cattle. Edited by Walter J. Gibbons, California: Amer. Vet. Publication, pp. 748-749.

- Cox, D. H. (1957). Isolation and identification of strychnine and other alkaloids in veterinary toxicology. *Amer. J. Vet. Res.*, 18:929-931.
- Durrel, L. W., and C. Aggio (1907). Poisonous and injurious plants of Colorado. *Expt. Sta. Bull.* 412-A, Colorado A and M College, Fort Collins. Quoted by Smith and Jones, *Veter. pathology* (1961), loc. cit.
- Done, J. T. (1957). Polio-encephalomalacia in swine. *Vet. Rec.*, 69:134.
- Epstein, H. (1955). Phylogenetic significance of spina bifida in zebu cattle. *Ind. Jour. of Vet. Sci.*, 25:313.
- Elliot, G. A. (1958). Focal necrosis in cerebral cortex of bovine. *Vet. Ext. Quar. Univ. Pa.*, No. 150:75-92. Abstracted from *Vet. Bull.*, para. 2995 (1958).
- Formston, C., and E. W. Jones (1956). A spastic form of lameness in freisian cattle. *Vet. Rec.*, 68:624-627.
- Gibbons, W. J. (1963). Diseases of cattle, Second edition. *Amer. Vet. Publications, California*, p. 261.
- Gordon, R. N. (1956). Snake bite and poisonous snakes. *Aust. Vet. Jour.*, 34:216.
- Goss, L. T., and F. E. Hull (1939). Spina bifida calf. *Cornel. Vet.*, 29:239.
- Gregory, P. W., S. W. Mead, and W. M. Regan (1944). Hereditary congenital lethal spasms in jersey cattle. *Jour. Hered.*, 35:(7)195.
- Hartley, W. J., and J. C. Kater (1959). Polioencephalomalacia in sheep. *N. Z. Vet. Jour.*, 7:75-80.
- Helve, A. (1958). Observations on the frequency of neurinoma in Finland 1952-1956. *Proc. VIII. Nord. Vet. Congr., Helsinki*, p. 244. Abstracted from *Vet. Bull.*, para. 3895 (1959).
- Howel, Mc. C. (1961). Polioencephalomalacia in calves. *Vet. Record*, 73: 1165-1167.
- Hulland, T. J. (1956). Cerebellar ataxia in calves. *Canad. Jour. of Comp. Med.*, 21:72-76.
- Innes, J. R. M., and L. Z. Saunders (1957). Advances in veterinary science. Vol. III, p. 43.
- Innes, J. R. M., Dorothy Russel, and A. J. Wilsdon (1940). Familial cerebellar hypoplasia and degeneration in hereford calves. *Jour. Path. and Bact.*, 50:455-461.

- Innes, J. R. M., and W. Plowright (1955). Focal symmetrical polymyalacia of sheep in Kenya. *J. of Neuro. Path.*, 14:185-197.
- Innes, J. R. M., and L. Z. Saunders (1962). *Comparative neuropathology*, 1962 edition. New York and London: Academic Press, loc. cit.
- Jensen, R., L. A. Griner, and O. R. Adams (1956). Polioencephalomalacia in cattle and sheep. *J. Amer. Vet. Med. Ass.*, 129:311-321.
- Johnson, K. R., D. L. Folwit, R. H. Ross, and J. W. Bartley (1958). Hereditary congenital ataxia in holstein-freisian calves. *Jour. Dai. Sci.*, 41:1371-1375.
- Johnson, L. A. Y., McGavin, and G. C. Simmons (1962). Diseases of bovine central nervous system in Southeast Queensland. *Aust. Vet. Jour.*, 38:521-527.
- Jolly, D. W. (1957). The toxicity of organic phosphates in insecticides. *Vet. Rec.*, 69:796.
- Kelser, E. M., and C. B. Knodt (1950). Synthesis of certain B vitamins in the digestive tract of dairy calves. *Jour. Dai. Sci.*, 33:381-383.
- Marsh, C. D., A. B. Dawson, and H. Marsh (1916). Larkspur poisoning in livestock. *Bull. 365*, U. S. Dept. of Agri. Quoted by Smith and Jones, loc. cit.
- Marigold, D. (1958). Neurofibromatosis in cows progress. *Vet. Torino*, 12: 176. Abstracted from *Vet. Bull.*, para. 835 (1958).
- McConnell (1946). Effects of lightning. *N. Amer. Vet.*, 27:220.
- McDonald, I. W. A. (1947). A staggers syndrome in sheep and cattle. *Aust. Vet. Jour.*, 18:251.
- McFarland, L. Z. (1959). Spina bifida with meningocele in cattle. *Jour. Amer. Vet. Med. Ass.*, 134:32.
- McGirr, J. L. (1956). Present day toxicity problems and review. *Vet. Rec.*, 68:902-909.
- Mellenby, E. (1938). Vitamin A deficiency. *Jour. of Physiol.*, 93:42.
- _____. (1939). Vitamin A deficiency. *Jour. of Physiol.*, 96:36.
- _____. (1941). Vitamin A deficiency. *Jour. of Physiol.*, 102:467.
- Moore, L. A., E. T. Hallman, and L. B. Sholl (1938). Cardiovascular and other lesions in calves fed on diet low in magnesium. *Arch. Path.*, 26:820-838. Quoted by Smith and Jones, p. 686.

- Moore, L. A., C. F. Hoffman, and C. W. Duncan (1935). Vitamin A deficiency in calves--eye lesions. *Jour. of Nutrition*, 9:533.
- Motten, S., and J. Penzance (1930). Symposium on lightning. *Vet. Record*, 8:(42)901-906.
- Moulux, A. W., and C. L. Davis (1953). Multiple Schwannomas in cattle. *Amer. J. Vet. Res.*, 14:449-509.
- Nes, N. (1959). Spina bifida accompanied by muscle contractures and other defects in cattle. *Nord. Vet. Med.*, 11:33-54.
- Nocklods, C. (1896). Poisoning by locoweed. *Amer. Vet. Res.*, 20:509. Quoted by Smith and Jones, p. 656.
- Pack, G. T. (1958). A clinical treatise of soft tissues. New York, p. 576. Quoted by Smith and Jones.
- Peterson, J. E. (1963). Squamous cell carcinoma of leptomeninges of brain in a cow. *Jour. Comp. Path.*, 73:163.
- Phillip, J., and M. C. Everny (1951). Accidental poisoning of dairy calves by Benzene hexachloride. *Cornell Vet.*, 41:295-299.
- Roberts, S. J. (1953). A spastic syndrome in cattle. *Cornel. Vet.*, 43:383-388.
- Saunders, L. Z., J. D. Sweet, S. S. Martin, F. H. Fox, and M. A. Fincher (1952). A hereditary congenital ataxia in jersey calves. *Cornel. Vet.*, 42:559-591.
- Schwartzkopf, D. (1888). Locoweed poisoning. *Amer. Vet. Rev.*, 12:160-162. Quoted by Smith and Jones, p. 656.
- Sholl, L. B., E. E. Sales, and R. Lagham (1939). Three cases of cerebellar agenesis. *Jour. Amer. Vet. Med. Assoc.*, 95:230.
- Simon, J., and R. L. Brewer (1963). Multiple neurofibromatosis in a cow and calf. *Jour. Amer. Vet. Med. Assoc.*, 142:1102-1104.
- _____ (1963). Multiple neurofibromatosis in a cow and calf. *Jour. Amer. Vet. Med. Assoc.*, 142:1388-1389.
- Smith, H. H., and T. C. Jones (1961). *Veterinary pathology*. Philadelphia: Lea and Febiger.
- Sullivan, D. J., and W. H. Anderson (1958). Tumors of bovine acoustic nerve. *Amer. Jour. of Vet. Res.*, 19:848-852.
- Teternik, K. et al. (1960). (In Russian). Abstracted from *Vet. Bull.*, para., 3648 (1960).

- Tommasini, A., and Marcota (1939). Meningioma in cattle. *Acta. Med. Vet. Napoli S.*, pp. 429-447. Abstracted from *Vet. Bull.*, para. 1912 (1960).
- Terlecki, S., and L. M. Markson (1961). Cerebrocortical necrosis in cattle and sheep. *Vet. Rec.*, 73:23-27.
- Terlecki, S., L. M. Markson, J. B. Spence, A. J. Stevens, C. N. Saunders, and A. H. Harris. (1961). Cerebrocortical necrosis in cattle and sheep. *Vet. Rec.*, 73:33-34.
- Udall, D. H. (1954). *The practice of veterinary medicine*. Ithaca, New York.
- Wiese, A. C., B. C. Johnson, and W. B. Neveus (1946). Biotin deficiency in dairy calf. *Proc. Soc. Expt. Biol. and Med.*, 63. Quoted by S. H. Morrison and C. F. Whitehair, "Diseases of Cattle," 1950.
- Williams, W. L., and J. L. Frost (1939). Subdural hydrocephalus in a calf. *Cornel. Vet.*, 28:340-345.
- Wright, G. P. (1955). *Mechanisms of microbial pathogenesis*. London: Cambridge University Press, pp. 78-102. Quoted by Blood and Henderson, p. 425.
- Young, Stuart (1962). Hypomyelinogenesis congenita (cerebellar ataxia) in angus shorthorn calves. *Cornell Vet.*, 52:84-93.

A COMPREHENSIVE STUDY OF NONINFECTIOUS NEUROLOGICAL
DISORDERS OF BOVINE

by

G. NARASIMHA MURTY

B. V. Sc., Madras University, 1943

AN ABSTRACT OF A MASTER'S REPORT

submitted in partial fulfillment of the

requirements for the degree

MASTER OF SCIENCE

Department of Medicine and Surgery

KANSAS STATE UNIVERSITY
Manhattan, Kansas

1964

A comprehensive study of the noninfectious neurological disorders of bovine has been made under the following classifications of

- (1) Congenital and/or hereditary defects,
- (2) Environmental and extrinsic factors,
- (3) Intrinsic factors,
- (4) Nutritional deficiencies,
- (5) Toxic plants and chemicals, and
- (6) Disorders of unknown etiology.

The available literature on the congenital and/or hereditary defects was very limited and most of the reports have been published during the past decade. Identical diseases are published under different titles. Some of the literature published on certain diseases, is incomplete.

The following conditions have been discussed under the hereditary defects, some of which are congenital defects also: cerebellar hypoplasia, congenital ataxia, cerebellar agenesis, spastic paresis, congenital posterior paralysis in calves, spastic syndrome in cattle, epilepsy, spina bifida, hydrocephalus and bulldog calves.

The conditions (1) arthrogryposis and hydranencephaly, and (2) neuronopathy and pseudolipidiosis have been dealt with under the disorders of unknown etiology even though they can be classified under congenital defects.

A complete description of the conditions, their familial and hereditary relationships, clinical signs and lesions have been described.

Under the extrinsic factors--the influence of environmental factors on animal body has been discussed.

The etiology, clinical signs, lesions have been discussed under the following conditions: (1) lightning stroke and electrocution, (2) heat

stroke, heat exhaustion, heat cramps, dehydration exhaustion, sun stroke, trauma, allergic encephalitis.

Under nutritional deficiencies, symptoms and lesions of vitamin and mineral deficiency diseases which manifest nervous disorders have been discussed.

The toxic effects produced by many of the poisonous plants have been described. The toxic principle contained in the plant and its mode of action on the nervous system, and the lesions produced have been described.

Many more toxic plants require investigations regarding their active principles, their toxicity levels and their pathogenicity. Chemical poisons which affect the nervous system, have been described. The toxic levels, mode of action on nervous tissue, clinical symptoms, and pathological findings are furnished.

Lastly the noninfectious diseases of nervous system whose etiology is not known such as polioencephalomalacia, neuronopathy and pseudolipidosis, and arthrogryposis and hydranencephaly have been described.

The noninfectious nervous disorders are generally sporadic and frequently escape investigation by veterinary pathologists.

Further investigations into the disease incidence, hereditary dispositions, clinical signs, and pathological findings are necessary.

An extensive review of all literature available is made and the conditions are discussed in detail.